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Conceptual Barriers to Interdisciplinary Communication

When Does Ambiguity Matter?[1]

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We examine three cases in which different conceptualizations of the same object of research have existed alongside one another. The first of these cases features the Mendelian and molecular identities of the gene. We show that these identities exist alongside one another in genetics and play complementary roles. In this case, ambiguity in a key theoretical construct has not led to misunderstanding but has promoted a productive slippage of meaning. We argue that is because of the creation of "boundary objects" (Star & Griesemer, 1989) shared between these different research traditions. Boundary objects, such as genetic loci and specific sequences of DNA nucleotides, contain features that play roles in two different scientific contexts and therefore facilitate the communication

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between them, despite them having different, or at least only partially overlapping, meanings in both fields. In our second case, we contrast two ways of thinking about genes found in different fields studying the genetic basis of behavior. We show that this has historically been a barrier to communication between those fields but that recent technical developments have promoted better communication. We argue that those developments have done so by creating boundary objects that facilitate this communication. Our third case looks at some disputes about behavioral development centered on the ambiguous and contested concept of innateness. We offer some tentative thoughts on how the intractability of these debates may reflect a paucity of functional boundary objects. In the first two of the three case studies, we draw extensively on arguments developed in our forthcoming book *Genetics and Philosophy* (Griffiths & Stotz, 2013), arguments that we can reproduce only in a much abbreviated form. We use the conclusions drawn there to reflect on the question of interdisciplinary communication. For those interested in the full arguments for these conclusions, we recommend consulting the book.

Mendelian and Molecular Genetics

In this section, we outline the Mendelian and molecular conceptions of the gene and show that they coexist in biology even today. The term *gene* is understood in two different ways by investigators, sometimes even by the same investigator at different points in a single research project. The flexibility of the gene concept, which allowed the coexistence of multiple conceptualisations of the gene, has generally been productive rather than problematic, as many authors have noted (Beurton, Falk, & Rheinberger, 2000; Falk, 1984, 1986; Griffiths & Stotz, 2013; Stanford & Kitcher, 2000; Waters, 1994). Our discussion illuminates how such slippage of meaning plays a productive role.

The Mendelian Gene and the Molecular Gene

The gene of classical Mendelian genetics—the research tradition that flourished in the first half of the 20th century, before the advent of molecular biology—had a very distinctive status. It was not an observable entity, but it was more than an unobservable entity postulated to explain the data. The gene was a tool for predicting and explaining the outcome of breeding one organism with another. Mendelian genetics calculates the results of breeding organisms using their genotypes, not merely their phenotypes. The relationship between parent and offspring phenotypes is mediated by relationships between genotypes. So the gene was not merely postulated to explain the success of Mendelian genetics; it played a key role in that successful scientific activity.

Geneticists naturally hoped that the gene would be shown to exist as a physical reality within the cells of the organism. But the special role of the gene in the practice of Mendelian genetics meant that the gene would remain an important and legitimate idea even if this did not work out. The leading classical geneticist T. H. Morgan remarked in his Nobel Prize acceptance speech, "At the level at which the genetic experiments lie, it does not make the slightest difference whether the gene is a hypothetical unit, or whether the gene is a material particle" (Morgan, 1934). One could interpret Morgan this way: If genes are not material particles, then they must be something like centres of mass in physics. When two bodies act on one another, for example by being at the two ends of a lever, their masses are distributed throughout each body. But when we calculate how the bodies will affect one another, the whole mass is assigned to a single, infinitesimal point—the centre of mass. The centre of mass is not a material particle, but every object nevertheless has a centre of mass. In the same way, even if there were no straightforward physical particles corresponding to genes, they would still exist in the calculus of heredity.

The historian of genetics Raphael Falk (1984, 2009) has summed up this situation by saying that the gene of classical Mendelian genetics had two separate "identities." One identity was as a hypothetical material entity, and some genetic research was directed to confirming the existence of these entities and finding out more about them. But the gene had a second, and more important, identity as an instrument used to do biology. The future development of genetics was the result of the interplay between these two identities.

The practice of classical genetics eventually led to the emergence of a new, molecular conception of the gene. This occurred because in the decades after the discovery of DNA the material identity of the gene became the most prominent, not because of dissatisfaction with the Mendelian paradigm. Rather, the Mendelian paradigm—the core ideas of Mendelian genetics and the practice of genetic analysis—was not overthrown but persists even today.

The rise of molecular biology was associated with a new conception of the gene as a distinctive molecular structure in the DNA sequence. This is the gene presented in biology textbooks today. Each gene has a "promoter region" that acts as a signal to the machinery that transcribes the DNA into RNA. This is followed by an "open reading frame," a series of nucleotides that correspond to the series of RNA codons that specify the amino acids in a protein, plus a "start codon" and a "stop codon" that act as signals to the respective machinery that transcribes the DNA into RNA and translates the RNA into protein. The gene of molecular biology is the linear image of a gene product in the DNA.[2] The linear correspondence between DNA and its products was at the heart of the vision of the gene advanced by Francis Crick as early as 1958, but its epistemological significance was brought to philosophical attention by C. Kenneth Waters (1994). Linear correspondences between nucleic acid sequences and other molecules are fundamental to

[2]We owe this expression to Rob D. Knight.

biologists' ability to identify and manipulate those molecules. That molecular genes are sequences that have a linear correspondence to their products (via DNA replication, DNA transcription, and RNA translation) is the key to the practical utility of the molecular gene concept in research and in biotechnology. This is true despite the extent to which transcriptional and posttranscriptional processes can distort this relationship in multicellular organisms (Djebali, Davis, et al., 2012; Djebali, Lagarde, et al., 2012; Gerstein et al., 2007; Griffiths & Stotz, 2013).

The Explanatory Roles of Genes in the Two Research Traditions

The emergence of the classical molecular gene appears to be a successful example of the research strategy of identifying a causal role and seeking the concrete occupant of that role. In this case, the role was that of the Mendelian gene, something whose distinctive pattern of transmission from one generation to the next explains the phenomena of heredity. We might suppose that the occupant of that role is the molecular gene—an open reading frame with an adjacent promoter region. But this turns out to be too simple.

The role-occupant framework (Lewis, 1966) starts from the observation that some concepts can be analysed in terms of the causes and effects of the thing being conceptualised, or its "causal role." Lightning was originally known only as something that causes bright flashes in the sky during thunderstorms and whose destructive effects we see as lightning strikes. When it was shown that the flashes and destructive strikes are the effects of atmospheric electrostatic discharges, it followed necessarily that lightning is atmospheric electrostatic discharge. Now, the gene was certainly originally identified by the causal role it played—it was the cause of Mendelian patterns of inheritance. Later, it was discovered that this causal role was played by pieces of DNA passing from parent to offspring. So it follows necessarily that these pieces of DNA are Mendelian genes.

This framework seems at first glance to provide a good model of what happened in the transition from Mendelian to molecular genetics: With the unravelling of the genetic code and of the basic processes of transcription and translation in the 1960s, the two identities of the gene in classical genetics, the instrumental Mendelian and the hypothetical material, converged on a single identity—the molecular gene. Looked at more closely, however, the theoretical role of the gene had been significantly revised so as to take account of findings about the material gene.

In classical Mendelian genetics, the gene played three theoretical roles. It was the unit of mutation—changes in genes give rise to new, mutant alleles[3]

[3]The "alleles" of a gene are the different forms of a single gene. In diploid organisms such as humans, each individual has two copies of each gene. These may be the same allele or different alleles.

of the same gene. It was also the unit of recombination. Crossover between chromosomes either separates genes that were previously linked or links genes that were previously inherited independently. Finally, the gene was the unit of function. The genotype that interacts with the environment to produce the phenotype is a collection of genes, and any effect of genotype on phenotype can be traced back to some gene or combination of genes. It was natural to project these ideas from Mendelian genetics onto the gene as a hypothetical material entity and to expect that the material gene would be a unit of mutation, of recombination, and of genetic function. But the new, molecular concept of the gene that emerged in the 1960s did not live up to that expectation.

A mutation is something that can happen to any stretch of the DNA molecule, not only to genetically meaningful units such as molecular genes. Recombination is a highly regulated process involving chromosomes and an accompanying cast of enzymes. In analysing this process, there is no need to divide the DNA itself into "units of recombination": Recombination can occur within a molecular gene so that one part of one allele is recombined with the complementary part from its sister allele, as well as occurring between whole alleles, and it can occur between segments of DNA that are not molecular genes at all. So the new conception of the molecular gene was one in which the gene is only the unit of function and not the unit of mutation or recombination, and with a slightly changed function at that.

The concept of the molecular gene applies only to sequences that have a structure something like that described above and that act as the template for making a gene product. But in humans, for example, only 1% to 2% of the DNA consists of structures used to make proteins. There are many segments of chromosome that have some effect on the phenotype and hence behave as Mendelian alleles but do not count as genes under the new molecular conception. Any difference in the sequence of DNA that causes a difference in phenotype will function as a Mendelian allele, but it need not be an allele of any molecular gene.

It would be foolish to redefine *allele* so as to restrict the term to alleles of molecular genes, because the fact that these other sequences are behaving as Mendelian alleles is not something to gloss over. If there is an inherited cause of phenotypic differences, we want to know about it! Conversely, the molecular gene cannot be redefined as any piece of DNA that can act as a Mendelian gene, because this would render it unsuitable for the purpose for which it is used in molecular biology—namely, to identify sequences that have a linear correspondence to the biomolecules made from the DNA.

These observations would be a mere quibble if as a matter of fact the pieces of DNA picked out by the instrumental, Mendelian conception of the gene were always sequences that are also genes according to the molecular conception of the gene. But this is not the case. There are now known to be many other ways DNA sequences can play a role in the development of phenotypes besides acting as linear templates for the synthesis of biomolecules. When one of these other pieces of DNA comes in two or more forms

with different phenotypic effects, they will behave as Mendelian alleles and can be investigated via genetic analysis. Even if they are not called genes, they are treated as (Mendelian) genes, and sometimes they *are* called genes but only when speaking in an appropriate context. Such is the flexibility of scientific language. For example, when a medical geneticist is seeking the "genes for" a disorder, she is looking for Mendelian alleles: sections of chromosome whose inheritance explains the phenotypic differences observed in patients. Translated into molecular terms, these sequences may turn out not to be molecular genes but segments of DNA that fulfil other, regulatory functions.

A clear example of the continuing coexistence of the Mendelian and molecular identities of the gene comes from studies of the gene *Lmbr1* in the mouse and its homologue[4] on human chromosome seven (Lettice et al., 2002). This locus is known to house an allele that produces abnormal limb development in both mice and humans. But further molecular analysis of that locus shows that the molecular gene within which the mutation is located is not a molecular gene that plays a role in the development of these abnormalities. Instead, there is a sequence embedded in a noncoding stretch within that gene that acts to regulate the gene "*sonic hedgehog*" (*shh*). The gene *shh* is located around a million DNA nucleotides away on the same chromosome and is known to be important in limb development. The regulatory sequence at the original locus is called an "enhancer" in molecular genetics, not a gene, since it does not code for a product. It is not a functional component of the molecular gene within which it is physically located, since this mutation doesn't affect the product of that gene or the posttranscriptional processing of that product. But this regulatory sequence is the Mendelian allele for the abnormal limb development. Conversely, *shh* is a paradigmatic molecular gene, but there exists no allele of *shh* that causes (is the Mendelian allele for) this kind of abnormal limb development. Instead, in one experimental context—that of hunting for the mutation responsible for the phenotype—the idea of gene assumes its Mendelian identity, while in the other context—that of analysing the DNA sequence—the idea of gene assumes its molecular identity. In many cases, these two identities of the gene converge on the same sequence of DNA, but sometimes they do not (for a more extended analysis of this issue, see Weber, 2005, pp. 215–233).

So the relationship between Mendelian and molecular conceptions of the gene defies at least the simplest form of role-occupant analysis (for a more adequate treatment, see Stanford & Kitcher, 2000). Biologists were looking for the physical occupant of the role of the Mendelian gene. But what they found, and what molecular geneticists call a gene, occupies only part of that role, and the original role remains important, too. This complex situation helps explain the long-running controversy in philosophy of science about

[4]The two genes are derived from a common ancestor, rather than evolved independently.

whether Mendelian genetics has been reduced to molecular biology (for a summary of this debate, see Schaffner, 1993; and for a similar diagnosis, Weber, 2005). One clear sense in which Mendelian genetics does not reduce to molecular genetics is that it continues to exist alongside molecular genetics as another, complementary way of thinking about DNA. Molecular biology enriched genetics with a new way of thinking about genes, and biologists today have two valid ways of thinking about genes. They move smoothly between these two contextually activated representations of genes as they move from one research context to another (Griffiths & Stotz, 2013).

The Boundary Objects Shared by Mendelian and Molecular Genetics

Biologists seem able to move smoothly between these two representations according to the context in which they find themselves. We suggest that this is possible because of the rich variety of "boundary objects" (Star & Griesemer, 1989) that exist in both contexts. Star and Griesemer introduce the idea of boundary objects as follows:

> Boundary objects are objects which are both plastic enough to adapt to local needs and constraints of the several parties employing them, yet robust enough to maintain a common identity across sites. They are weakly structured in common use, and become strongly structured in individual-site use. They may be abstract or concrete. They have different meanings in different social worlds but their structure is common enough to more than one world to make them recognizable, a means of translation. The creation and management of boundary objects is key in developing and maintaining coherence across intersecting social worlds. (p. 393)

Star and Griesemer's (1989) paper outlines an approach to case studies in history of science that explains how scientists manage to cooperate despite heterogeneity in their scientific and social fields. In their original case study, they analysed the collaboration between professionals, administrators, patrons, and amateurs connected to a research natural history museum. Here, we are interested in communication between different areas of genetics (in the next two sections, we will look at two further examples of communication between scientists from different research traditions). The boundary objects that feature in genetics may be abstract, such as genetic loci, or concrete, such as a specific mutant strain of *Drosophila* that can pass from one lab to another. In either case, these boundary objects exist in both research contexts and therefore allow the integration of research conducted with different conceptualizations of the object of that research.

A good example of boundary objects in genetics is the "loci" that genes occupy on chromosomes. Genetic loci are like the latitude and longitude

system used in terrestrial navigation, a highly stable practice of identifying location supported by widely agreed-on procedures that are updated as better methods become available. The investigations into limb abnormalities described above involved the locus 7q36, the 36th band from the centromere on the long arm (q) of chromosome 7. The staining procedures needed to make such bands on the chromosome visible were historically important, but the regions named in this way can now be accessed via genome databases containing a standardized representation of the actual DNA sequence. Loci are thus not tied to any one experimental technique. The same loci are used in both Mendelian and molecular genetics. In an instructive exercise, Marcel Weber (2005, pp. 215–233) compared descriptions of certain loci in *Drosophila* made using classical genetic methods and later descriptions of the same loci made using molecular methods. These descriptions often agree in the number of genes at the locus and their roles, but sometimes they do not, something we have already shown in our example of limb abnormalities. But, however complex the relationship between the genes identified by Mendelian methods and those identified by molecular methods, the shared loci that both disciplines recognise provide common ground—almost literally—on which to explore and resolve those differences.

In this section, we have described two conceptions of the gene that play a role in contemporary biology. A third will be described in the next section, and we have described yet others elsewhere (Griffiths & Stotz, 2007, 2013). We suggest that the availability of well-characterized lines of organisms, well-characterized mutations, practices of chromosome mapping, and, later on, the availability of DNA sequence data—all of which can act as "boundary objects" between different research contexts—explains how genetics has been so successful with such a multifaceted and contextual concept as that of the gene at its heart.

Genes and Behavior

In this section, we will introduce another ambiguity in the concept of the gene but one that has given rise to greater problems for interdisciplinary communication. We will suggest that this was due to an inability to create boundary objects that would have allowed communication without a shared conceptualisation of the gene, a situation that is now changing.

The Mendelian and the Abstract Developmental Gene

There are two very different ways responsibility for a behavioral difference can be sheeted home to a genetic difference. These involve two identities of the gene, the familiar Mendelian allele and another identity that we call the "abstract developmental gene." The ambiguities introduced by these

different ways of thinking about genes, and the different conceptions of gene action that accompany them, have led to severe miscommunication between scientists from different disciplines that study the genetic basis of behavior.

Traditional behavioral genetics was the application of Mendelian genetic analysis and quantitative genetics to behavioral phenotypes. In human behavioral genetics, quantitative genetic methods predominated. Quantitative genetics was developed to integrate Mendelian genetics with statistical methods for studying heredity. The most familiar result of quantitative genetics is a figure for the *heritability* of a phenotypic trait. The heritability of a trait is used to estimate the extent to which the phenotypic differences between individuals in a population can be explained by genetic differences between those individuals. In human behavioral genetics, where it is not possible to conduct controlled breeding experiments, heritabilities are inferred from observations of the correlations between relatives. For example, one kind of "twin study" uses the correlations between monozygotic twin pairs, who share all their genes (meaning all their Mendelian alleles), and dizygotic twin pairs, who share only half their genes. If the monozygotic twins resemble each other more closely than do the dizygotic twins, then, all other things being equal, this can be attributed to their greater proportion of shared genes.

Behavior genetics has always been controversial because of concerns about its social and political implications. This has tended to draw attention away from more substantial scientific criticisms, but in fact there have been many of these. Some of the strongest criticism of behavioral genetics has come from scientists who study the development of behavior, a field known as developmental psychobiology. Developmental psychobiologists have historically been scathing about quantitative genetic approaches, arguing that they do not yield any genuine scientific insight into the genetic basis of behavior (Griffiths & Tabery, 2008; Tabery & Griffiths, 2010).

Behavior geneticists, and quantitative geneticists more generally, conceptualize genes as Mendelian alleles. While they have not typically identified specific Mendelian alleles for complex human behaviors, their study designs and statistical models deal with the consequences of differences in the proportion of shared Mendelian alleles between individuals. In contrast, developmental psychobiologists conceptualize genes as determinants of the value of a developmental variable in the context of a larger system (we will refer to these as "abstract developmental genes"). In many instances, we think that both sides would pick out the same specific DNA sequence elements if they had sufficient information about the molecular basis of a trait. But until very recently, that information has not been available. Genes have existed for both communities only in the conceptual foundations of their methods, rather than being grounded in specific sequences of DNA that could have functioned as boundary objects that would have allowed the two communities to transcend their different ways of thinking about "genes."

Traditional work in developmental psychobiology investigates how normal development at each stage of the life cycle depends on the interaction of

the organism with specific features of the environment. This work typically involved experimentation under controlled conditions rather than the observational analysis of natural populations. Recent work in developmental psychobiology has begun to link factors in these models to the expression of specific sequences in the genome. However, for most of the history of this research tradition, it has not been possible to experimentally manipulate genetic factors of a developmental system in the same way as environmental factors. Although developmental psychobiologists conceived of genes as mechanistic causes of development, the lack of direct access to these causes led them to appear in an extremely abstract form, as the hypothesised determinants of the value of certain parameters or variables in the model.

This "abstract developmental" conception of the gene can be traced back to the attempts of embryologists to integrate genetics into their discipline in the 1930s. If it is assumed that the biochemical processes that construct phenotypes are the result of gene action, then some or all of the factors in a developmental model can be labelled as "genes." Julian Huxley (1932/1972) speaks of "rate genes" determining the value of variables in his models of relative growth. These hypothetical genes have no empirical foundation besides the model itself. The same abstract conception of the gene features in the famous models of "developmental canalization" due to C. H. Waddington (1940, 1957).

The Causal Roles of Genes in the Two Research Traditions

Mendelian alleles and abstract developmental genes are two legitimate ways to think about DNA sequences in two very different research contexts. But the explanatory roles the "gene" plays in those two contexts are very different. An abstract developmental gene can explain a phenotype only via the mediation of many other developmental factors. In contrast, the Mendelian allele for a phenotypic difference explains that difference without the need for explicit reference to other developmental factors. The abstract developmental gene has no identity apart from its role in a developmental model. In a model intended as an actual characterization of a developmental process, the introduction of specific "genes" is justified by reference to the ability of the model as a whole to explain the effects of manipulations of its variables. So explaining the presence of a phenotype by reference to the presence of a gene means drawing attention to how that genetic factor interacts with the other factors. The same is true of phenotypic differences, which are explained by reference to how a genetic difference ramifies through the system (Griffiths & Tabery, 2008; Tabery & Griffiths, 2010). Developmental psychobiologists seek to "explain" phenotypes in the sense characterised by mechanist theories of explanation in the philosophy of science (Griffiths & Stotz, 2013): The fact that development produces an outcome is explained by showing that the specific ways the parts of that system are arranged, and the behavior of those parts in accordance with standard physical laws, lead to the phenotypic outcome.

Explanations of phenotypes in terms of the presence of Mendelian alleles do not share these features. The presence of an allele (or of different alleles) explains the presence of the associated phenotype (or a phenotypic difference) because of a statistical association between alleles and phenotypes in a pedigree or a population.[5] The epistemological value of this relationship derives precisely from the fact that it applies robustly across the actual distributions of developmental factors in the population from which it is derived and in which it can be legitimately extrapolated, or that it applies often enough for the average effect produced to be useful knowledge. Thus, the abstract developmental gene explains by reference to the causal structure of the developmental system, whilst the Mendelian allele explains by importing statistical information about specific alleles and phenotypes from some reference class. Explanation in this context is best understood not as providing a mechanism but as causal explanation in James Woodward's sense (Waters, 2007; Woodward, 2010): The cause is a variable that can be used to manipulate another variable, the effect, and this relationship is robust across a certain range of values of other variables.

Thus, from the perspective of the abstract developmental gene, it makes no sense to explain the presence of a phenotype (or difference) by alluding to the presence of a particular gene in the absence of any understanding of its role in a broader developmental system. From this perspective, the fact that a gene has a specific phenotypic effect raises the question of why it has had that effect rather than another effect it might have had if other factors had been different. The frequent claim by developmental psychobiologists that the mere presence of a gene cannot in itself explain the presence of a phenotype reflects this conception of how genes explain phenotypes.

Conversely, if genes are Mendelian alleles, then it seems unreasonable to demand knowledge about how a gene interacts with other genes and with the environment before accepting an explanation that cites the presence of this allele as the "actual difference maker" between one phenotype and another (Waters, 2007). If the organism or organisms whose phenotypes are to be explained have been drawn from a suitable reference class, then those other factors will not make a difference (Griffiths & Tabery, 2008; Tabery & Griffiths, 2010).

Hence, an important aspect of the disagreement between behavioral geneticists and developmental psychobiologists is over the scientific relevance of factors that do not vary in nature. Behavioral geneticists argued that developmental factors that do not account for any of the actual variance seen in populations are irrelevant to the explanation of *phenotypic differences* seen in those populations. In contrast, the causal–mechanistic study of

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[5]Note that we have switched back from considering quantitative genetic models to considering a simple Mendelian model in which a single genetic difference makes a phenotypic difference. But the same point applies when many genetic differences make small contributions to a phenotypic difference.

behavioral development has traditionally been concerned with the development of *species-typical phenotypes*, a feature it shares with most traditional developmental biology. The factors that do not vary are relevant because they are part of the developmental process in virtue of which those that do vary exert an influence on the phenotype. Because developmental psychobiology aims to characterize the causal mechanisms of development, it has no reason to privilege *actual* causes over *potential* causes (Waters, 2007).

One of the most dramatic misunderstandings that has arisen from these conceptual differences concerns "gene–environment interaction." In behavioral genetics, gene–environment interaction is understood as a statistical phenomenon that requires the introduction of an additional term into the analysis of variance. Part of the variance is a "main effect" of genetics (G); another part is a main effect of environment (E), but a further part is an "interaction effect" (G×E). In contrast, for developmental psychobiologists, interaction is not primarily a statistical phenomenon. It is the fact that genetic and environmental factors physically interact in the causal processes that give rise to phenotypes—that a single developmental mechanism has components that are genetic and components that are environmental.

James Tabery (2007) has labelled these two concepts of gene–environment interaction the "biometric" concept ($G\times E_B$) and the "developmental" concept ($G\times E_D$). The long-standing dispute between behavioral geneticists and their critics over gene–environment interaction is to a significant extent the result of their using these two different concepts of interaction, corresponding to the two different conceptions of the gene outlined above. Behavioral geneticists regularly detect large main effects for genes and fail to identify a high level of statistical interaction ($G\times E_B$) between genes and environment. For a behavioral geneticist, this translates, by definition, into low interaction. But for developmental psychobiologists, interaction is fundamentally a property of causal networks and $G\times E_B$ is only the statistical manifestation of actual causal relationships. Developmental psychobiologists claim that gene–environment interaction is ubiquitous despite the failure of behavioral geneticists to detect large statistical interactions (Gottlieb, 2003, p. 343). Michael Meaney (2001) writes that "phenotype emerges only from the interaction of gene and environment. The search for main effects is a fool's errand. In the context of modern molecular biology, it is a quest that is without credibility" (p. 51). Meaney is undoubtedly aware that substantial main effects *are* found in this context, so to understand what he means, we need to recognise that two different senses of interaction are in play. Behavioral geneticists have recognized this, but they have typically argued that the statistical sense is the important one and that the other sense amounts to little more than complexity worship:

> Unfortunately, discussions of genotype–environment interaction have often confused the population concept with that of individual

> development. It is important at the outset to distinguish genotype–environment interaction from what we shall call *interactionism*, the view that environmental and genetic threads in the fabric of behavior are so tightly interwoven that they are indistinguishable. (Plomin, DeFries, & Loehlin, 1977, p. 309)

But a far more sympathetic reading of "interactionism" is possible. Developmental psychobiologists think they have conclusive evidence for the highly interactive (GxE_D) nature of development: "From such systems will we derive main effects? I think not" (Meaney, 2001, p. 54). But, as we have noted, behavioral geneticists do extract substantial main effects from such systems. Scientists such as Meaney respond to this absence of statistical interaction (GxE_B) by looking for experimental interventions that will create statistical interaction corresponding to the causal interaction (GxE_D). This can often be done by exposing the organism to environments outside the normal range. In other cases, the lack of statistical interaction under normal conditions indicates that development is structured so as to render some outcomes insensitive to some range of environmental variation mechanisms such as redundancy, canalization, and feedback. In that case, experimentation on modified versions of the system itself may be required to disentangle these details of the mechanism.

It is sometimes argued that, while the causal sense of interaction is relevant to understanding how each individual develops, it is not relevant to understanding development at the population level—to explaining differences between individuals. But Tabery has argued convincingly that the developmental, causal sense of interaction *can* be coherently applied to individual differences in a population, and in recent years scientists have started to describe how differences arise from what Tabery (2009) calls "difference mechanisms."

Finding Boundary Objects for the Two Research Traditions

Traditional quantitative genetic methods in behavioral genetics are rapidly giving way to molecular methods. This seems to have created the grounds for a rapprochement between the two sides in this dispute. It has led to a greater appreciation by behavioral geneticists that the value of "gene hunting" is not to identify *the* cause of a behavioral difference but to find an entry point to the molecular pathways involved in the production of that difference (Hamer, 2002). Conversely, the effects of environmental interventions in developmental science are increasingly being analysed at the level of gene expression (Meaney & Szyf, 2005; Suomi, 2004). The ground on which the disputants are meeting is the actual sequence of DNA nucleotides. The ability to investigate actual DNA

sequences produces shared boundary objects that simultaneously make concrete both abstract developmental genes and the Mendelian alleles whose existence was inferred from quantitative genetic studies.[6] This is not to say that the two disciplines have converged on the molecular concept of the gene. Both abstract developmental genes and Mendelian alleles may be made concrete as DNA sequences that are not molecular genes, for the reasons described in the previous section. The critical convergence between the two approaches is less in their concept of the gene than in their methods of investigation and in the nature of the objects investigated—namely, DNA sequences and their expression levels. We suggest that this is another example of the scientific role of boundary objects (Star & Griesemer, 1989). DNA sequences and their expression levels are objects of investigation that can mediate between different intellectual contexts.

In the previous section, we encountered two alternative, contextually activated representations of the gene that coexist in biology, corresponding to two different ways of thinking about DNA grounded in two different kinds of scientific activity. We suggested that this led to productive slippage of meaning that facilitated scientific progress, rather than unproductive miscommunication, because of the abundance of boundary objects shared between the different contexts. In this section, we have seen two different representations of the gene built into the foundations of two very different research approaches to the genetic basis of behavior. The result was several decades of unproductive miscommunication. We have suggested that this situation will be—and is being—resolved by the construction of shared boundary objects due to the introduction of molecular methods in both disciplines. We do not suggest that students of the genetics of behavior will all come to conceptualize genes in the same ways but, rather, that disagreements will increasingly be perceived as empirical or conceptual, rather than as the result of sloppy thinking by one's opponents.

The Contested Concept of Innateness

Here we introduce a third and final example, one that is widely agreed to have led to long-standing debates that are "semantic" in the worst sense of the word. This is the concept of innateness, which lies at the heart of much of the debate over nature and nurture.

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[6]Searching for these alleles has created the so-called "missing heritability" problem. Current methods typically reveal a large number of loci, each of which accounts for a very small amount of variance and which collectively account for much less of the variance than is believed to be genetic on the basis of traditional quantitative genetic studies (see, e.g., Manolio et al., 2009).

Between Nativism and Anti-Nativism

It is a truism that the term *innate* is multiply ambiguous:

> At least six meanings are attached to the term: present at birth; a behavioral difference caused by a genetic difference; adapted over the course of evolution; unchanging throughout development; shared by all members of a species; and not learned. . . . Say what you mean (even if it uses a bit more space) rather than unintentionally confuse your readers by employing a word such as innate that carries so many different connotations. (Bateson, 1991, pp. 21–22)

In later work, Matteo Mameli and Patrick Bateson (2006) identified no fewer than 26 proposed definitions of *innate* from the scientific literature and judged 8 of these to be both genuinely independent definitions and separate, potentially valuable scientific constructs.

We and our collaborators have presented evidence that the idea of an innate trait is one expression of an implicit theory of the "natures" of living things shared by scientists and nonscientists alike (Griffiths, 2002; Griffiths, Machery, & Linquist, 2009; Linquist, Machery, Griffiths, & Stotz, 2011). Just as there are common-sense ideas about physical objects and the forces acting on them ("folk physics"; see, e.g., Clement, 1983), so there are common-sense ideas about biology. It is part of "folkbiology" (Atran, 1999) that some traits are expressions of the inner nature of animals and plants whilst other traits result from the influence of the environment. Echoing older critiques of the innateness concept in animal behavior research, we argue that folkbiology conflates the three issues of whether a trait is typical of the species, whether it is part of the design of the species (an adaptation), and whether its development is insensitive to the environment. Attempts to redefine innateness in a way that stresses just one of its aspects have been and will continue to be stymied by the fact that this multiply ambiguous concept of innateness is entrenched in the common-sense way of looking at the world.

The view that the ambiguity of the term *innate* obstructs scientific understanding has a long history in animal behavior research. At the heart of this critique is the claim that innateness conflates different biological questions and leads researchers to commit fallacies of ambiguity. For example, evidence that a trait is an adaptation is used to reach the conclusion that it is species typical, or vice versa. Evidence that a trait is an adaptation is used to infer that it is insensitive to environmental variation, or vice versa (for some recent examples, see Linquist et al., 2011, p. 445). When such inferences are laid out explicitly, it is clear that they do not follow without further evidence. However, if the discussion is conducted in terms of whether the trait is "innate"—a term that is used on different occasions to refer to each of these distinct, biological properties—it is easy to slide from one to the other.

The ambiguity of the innateness concept has not proved productive for the study of behavioral development. The study of innate behavior is not known for mutually supportive communication between different fields, each of which conceptualises the innate/acquired distinction somewhat differently. Instead, nativists[7] and anti-nativists are both convinced that they have a sophisticated vision of development that does justice to the complementary roles of gene and environment, and regard their opponents as confused (compare, for example, the diametrically opposed visions of how the nature/nurture dispute has been resolved in two very well-informed popular books, Gary Marcus's [2004] *The Birth of the Mind: How a Tiny Number of Genes Creates the Complexities of Human Thought* and David S. Moore's [2001] *The Dependent Gene: The Fallacy of "Nature vs. Nurture"*).

The Absence of Boundary Objects

Can this situation be explained by an absence of boundary objects that would allow the opponents' differing understandings to be reframed by some wider collective activity, such as shared investigative strategies? We tentatively suggest that this diagnosis may, indeed, be applicable here as well. Some of the fields in which nativism and anti-nativism are popular certainly use very different methods to investigate behavioral development. There is a strong strain of nativism in contemporary cognitive developmental psychology. Noam Chomsky's (1965) "language acquisition device" has served as an exemplar for research on the innate contributions to other psychological domains. Like the language acquisition device, other putatively innate features of the mind are thought to embody innate "knowledge" or innate "theories" about specific cognitive domains. For example, the eminent cognitive developmental psychologists Susan Carey and Elizabeth Spelke (1996) argue that children possess four domains of innate "core knowledge" that underlie much of their later cognitive development. These domains are "objects, agents, numbers and space" (p. 517; see also Carey, 2011). In theory, nativist developmental psychologists put a great deal of weight on "poverty of the stimulus arguments": The environment of the child does not contain the right stimuli for the child to acquire certain things, which must therefore be innate. However, collecting evidence for a poverty of the stimulus argument is demanding, and there is little evidence of poverty of the stimulus for many aspects of human psychology that have been labelled innate (Sterelny, 2003). Instead, many nativist claims rest on evidence that the putatively innate features are specific to one cognitive domain rather than another, that they emerge in a characteristic sequence in the development of the child, or that the same features are found in many human cultures. The last of these features speaks to the issue of whether the

[7]Nativism is the view that some aspect of mind or behavior is innate.

feature is species typical. The first two, and especially domain specificity, speak to the issue of whether the putatively innate feature serves a specific function and is plausibly a result of evolutionary design.

Anti-nativists are a more diverse group. Here we want to emphasise the opposition to nativism from the research tradition discussed in the previous section, developmental psychobiology. These researchers stress the dependence of all aspects of development on specific interactions with the environment.[8] A classic research exemplar is Celia Moore's (1984) work on penile reflexes in rats (see also Moore, 1992). She showed that the spinal cord nuclei of male rats differ from those of female rats in ways that allow the male to use his penis during copulation. These neural differences result from differences in gene expression in the developing spinal cord of the rat pup, which in turn result from differences in the licking of the genital area by the mother, which in turn result from the different composition of the urine of male pups. The ability of the male rat to use his penis during copulation is a species-typical trait and surely an adaptation, but the fact that it depends on very specific interaction with the environment is taken by developmental psychobiologists to show that it is not innate. This concern with dependence on the environment speaks to the aspect of the innateness concept according to which innate traits are insensitive to the developmental environment.[9] This is a contrast to the nativist research foci described above, which speak to the aspects of the innateness concept according to which innate traits are typical of the species and part of the design of the species.

Our argument at this point is tentative and exploratory. Our sketch of these two research traditions has been very brief, and more evidence and argument are needed to document the contrast we have suggested between them. If such a contrast does exist, however, there may be few shared boundary objects for these two traditions that would help them translate their differences into cooperation. There may be few concrete boundary objects because the two work on very different experimental systems. Nativist cognitive developmental psychology has a strong human focus. Developmental psychobiology has traditionally relied on experimental interventions that perturb the normal course of development, something that leads to an inevitable focus on animal models.[10] There may also be few

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[8]Good popular introductions to this research tradition, with a plethora of examples, are Bateson and Martin (1999) and Moore (2001). For a textbook treatment, see Michel and Moore (1995), and for a handbook approach, Hood, Halpern, Greenberg, and Lerner (2010).

[9]The same might be said of another major strand of anti-nativist thought, so-called "neural constructivism" (Elman et al., 1996).

[10]A notable exception to this generalisation is Esther Thelen and collaborators' "microgenesis" paradigm for the study of infant development (Thelen & Smith, 1994; Thelen & Ulrich, 1991).

abstract boundary objects. For example, the idea of a cognitive domain is important in nativist work on cognitive development, but cognitive domains do not constitute a shared ground in the way genetic loci do for different conceptions of the gene, since they cannot be anchored to a shared map of the brain in the same way genetic loci are anchored in shared representations of chromosomes.

Take-Home Messages

- In our first example of Mendelian and molecular genetics, we described how two alternative, contextually activated representations of the gene coexist in biology, corresponding to two different ways of thinking about DNA grounded in two different kinds of scientific activity. We suggested that this led to productive slippage of meaning rather than unproductive miscommunication because of the abundance of boundary objects shared between the different contexts.
- In our second example—research on the genetic basis of behavior in traditional, quantitative behavioral genetics and in developmental psychobiology—we described two different representations of the gene built into the foundations of these different forms of research regarding the genetic basis of behavior. The result was several decades of unproductive miscommunication. We suggested that this situation has been improved by the introduction of molecular methods in both disciplines, resulting in a sharing of boundary objects.
- In our third example, we examined disputes about behavioral development focused on the concept of innateness. We tentatively sketched some ways the inability to resolve these disputes might reflect an absence of boundary objects.
- The take-home message of these examples is a form of empiricism—although we hope not a naïve one. Scientists and science commentators frequently decry the vagueness or ambiguity of key theoretical constructs and suggest that disputes about such constructs are more semantic than empirical.[11] We would argue that the way forward in such cases is not, as is usually supposed, to seek a precise, stable conceptualisation of the subject matter. If we are correct, then the way to achieve mutual understanding, and to convert semantic disputes to empirical ones, is not to construct a conceptual straitjacket but to seek materials that can act as boundary objects and around which a thousand conceptual flowers may bloom.

[11]Another such dispute, to which one of us has contributed, concerns the concept of emotion in psychology (Griffiths, 2007).

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